Editorial

Modulation of Brain Criticality via Suppression of EEG Long-Range Temporal Correlations (LRTC) in a Closed-Loop Neurofeedback Stimulation

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PII: S1388-2457(16)30422-9
DOI: http://dx.doi.org/10.1016/j.clinph.2016.05.359
Reference: CLINPH 2007857

To appear in: *Clinical Neurophysiology*

Accepted Date: 28 May 2016

Modulation of Brain Criticality via Suppression of EEG Long-Range Temporal Correlations (LRTC) in a Closed-Loop Neurofeedback Stimulation

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Human behavioral and cognitive performance are beset by imperfections. Even in simple tasks, where a subject is instructed to tap with a finger at a fixed interval produce errors, no matter whether the tapping is cued by a metronome (Hennig et al., 2011) or not (Gilden et al., 1995). Fluctuations of tapping errors do not demonstrate a random variation between taps but show temporally correlated patterns that extend up to hundreds of seconds (Gilden et al., 1995). The error time series can be described via a power-law of the frequency spectrum defined by $P(f) \propto 1/f^{\beta}$, where P denotes the power of the frequency component f, while the scaling exponent β refers to the propagation of tapping errors (Gilden et al., 1995). The range of the observed β values lies around 1 suggesting that the time series describing the tapping errors have long-range temporal correlations (LRTCs) (Chen et al., 1997; Rangarajan and Ding, 2000; Hennig et al., 2011; Torre et al., 2011).

We know for many decades that psychological time series are not randomly clustered. For example, reaction-time and hit-rates in continuous performance tasks (CPT) are power-law auto-correlated within over hundreds of seconds time-windows (Chen et al., 2001; Gilden et al., 1995; Gilden, 2001; Helps et al., 2010; Ihlen and Vereijken, 2010; Monto et al., 2008; Thornton et al., 2005). Tasks tapping into other cognitive domains such as size estimation and detection of threshold stimuli also demonstrate power-law frequency scaling (Gilden et al., 1995; Gilden, 1997; Monto et al., 2008). Naturalistic man-made sequences outside the lab-oriented experimental paradigms like the fluctuations of loudness in speech and music show LRTCs (Voss and Clarke, 1975; Levitin et al., 2012). The biological origins and relevance of these dynamics, however, remain unclear (Farrell et al., 2006; Kello, 2010).

Similar to behavioural performance, the fluctuations of neuronal activity at various spatio-temporal scales exhibit scale-free dynamics governed by distributions of power-law. On long times scales of around 100 s, amplitude envelopes of spontaneous brain activity recorded with both magneto- and electro-encephalography (M/EEG) demonstrate both LRTCs and scale-free fluctuations (Linkenkaer-Hansen et al., 2001). These fluctuations of amplitude envelopes based on M/EEG reflect the underlying spontaneous human brain activity discovered via functional MRI (fMRI) and are defined by coherent activity described over slow fluctuations of blood oxygenation level-dependent (BOLD) signals among anatomical and functional distinct brain systems (Biswal et al., 1995; Raichle, 2001; Damoiseaux et al., 2006). The oscillation amplitudes are directly correlated with these BOLD fluctuations (Goldman et al., 2002; Leopold et al., 2003; Mantini et al., 2007; Sadaghiani et al., 2010; Scholvinck et al., 2010) while the coherent M/EEG related maps are closely related to the correlation maps derived from BOLD activity (Leopold et al., 2003; Brookes et al., 2011; de Pasquale et al., 2010; Nikouline et al., 2001). Moreover, BOLD signals also exhibit scale-free temporal dynamics (Suckling et al., 2008; Wink et al., 2008; He, 2011) and correlations in various spatiotemporal scales (Equiluz et al., 2005; Expert et al., 2011; Tagliazucchi et al., 2012). It is more than evident that scaling law of LRTCs is a unifying fundamental characteristic of spontaneous brain activity recorded with EEG, MEG and fMRI imaging methods (Chialvo, 2010; He et al., 2010).

In temporal scales from seconds to hundreds of seconds, the amplitude fluctuations and psychological dynamics of neuronal oscillations obey power-law distributions of LRTCs. In the millisecond temporal scale, neuronal activity encapsulates neuronal avalanches that also exhibit power-law distribution and lifetime distributions (Palva et al., 2013). Fractal properties of neuronal activity LRTCs and avalanches and also power-law scaling behaviour support that brain functions are near a critical state (Linkenkaer-Hansen et al., 2001; Chialvo, 2010; Beggs and Plenz, 2003; Plenz and Thiagarajan, 2007; Werner, 2010). Computational modelling studies validated that LRTCs and neuronal avalanches are coupled (Poil et al., 2008) and this relationship is supported from interactions of neuronal circuits in a critical regime (Chialvo, 2010). A study based on task and resting-state source reconstructed M/EEG recordings and behavioural responses demonstrated that behavioural scaling laws, LTRCs and neuronal avalanches are significantly correlated (Palva et al., 2012).

Scale free property characterizes self-similar processes which means that their properties are similar at every scale (Hardstone et al., 2012). When a complex system operates at a critical state then its characteristic dynamics presents a scale-free profile (Chialvo, 2010). A complex system functions at a critical regime when it demonstrates critical neural dynamics which are present when a system operates in the boundary between regularity and randomness (Hernandez-Urbina and Herrmann, 2016). When a complex system balances at this boundary then it is more flexible and it can achieve its maximum computational power (Shew et al., 2009; Kinouchi and Copelli, 2006), transmission capacity and optimal information storage (Shew et al., 2011). To quantitatively describe scale-free dynamics of a complex system that operates in a near critical state, the corresponding power-law scaling exponent of LRTCs can be estimated (Bak et al., 1987). The scaling exponents related with LRTCs denote the decay of auto-correlations, and by adopting the algorithmic approach of detrended fluctuation analysis (DFA), range between 0.5 to ~1, where 0.5 indicates a temporally uncorrelated time series (Peng et al., 2004).

Scaling exponents of LRTCs can be valuable predictors of behavioral dynamics (Palva et al., 2013; Smit et al., 2013) and have been suggested as biomarkers of many brain diseases (Linkenkaer-Hansen et al., 2005; Montez et al., 2009; Nikulin et al., 2012). LRTCs characterize the amplitude envelopes of human neuronal oscillatory dynamics in MEG and EEG (Linkenkaer-Hansen et al., 2001) and in intracranial recordings (Monto et al., 2007; Zhigalov et al., 2015).

We analyzed MEG spontaneous dynamic functional connectivity in both non-impaired and reading-disabled children via the notion of network metrics (global and local efficiency (GE/LE)) and by analyzing nodal network metric time series ($NMTS^{GE/LE}$) via DFA, and detected significantly lower values of scaling exponents for reading-disabled children compared to non-impaired readers over left temporo-parietal brain areas (Dimitriadis et al., 2013). Recently, analyzing spontaneous MEG source activity independently for amplitude and phase in carriers and non-carriers of APOE-e4 allele, we revealed significant group-differences on the scaling exponents based on the phase but not amplitude specifically in the β-band and γ-band in several regions of interest (Dimitriadis et al., 2016).

To discover how brain criticality can be modulated, it is important to examine how (ab)normal LRTCs are linked to human brain (dys)functions in relationship to cognition. This is an important step for designing novel therapeutic strategies for various brain disorders and diseases associated with LRTCs. For a complex system like the human brain to operate on a critical level, it is important to keep a balance between inhibition and excitation (Shew et al., 2009; Beggs and Timme, 2012) and to avoid super-critical and sub-critical states that are linked with extreme levels of neuronal excitation and inhibition, respectively.

Neurofeedback technologies have attracted growing interest from various fields of research and have been applied, *e.g.*, in the treatment of brain disorders such as the attention-deficit hyperactivity disorder (ADHD) (Arns et al., 2009), depression (Linden, 2014), Parkinson's disease (Subramanian et al., 2011), epilepsy (Strehl et al., 2014), and patients with severe neuromuscular disorders (Wolpaw et al., 2002). Additionally, neurofeedback has been adopted into brain computer interfaces (BCI), applications that gained popularity in video gaming and generally in digital entertainment (Kaplan et al., 2013). Nevertheless, neurofeedback is not yet widely acknowledged by the neuroscience community as a common research strategy in cognitive neuroscience due to technical and conceptual difficulties (Jensen et al., 2011).

Numerous studies focused on modulating the α-rhythm (8–12 Hz) via neurofeedback (for review, (Gruzelier, 2014a). An increase of power in α-rhythm has led to varied cognitive improvements like increased performance in working memory capacity in a digit span test (Escolano et al., 2011) and in a mental rotation task (Zoefel et al., 2011). Neurofeedback training focusing on α-rhythm has been reported to provide both affective and cognitive benefits like improved mood, intelligence, behavioral responses, like reaction time and sustained attention (Gruzelier, 2014a), which is a significant indicator that neurofeedback strategies might alter the functionality of neuronal processing. Endogenously, human perceptual performance can be improved via neurofeedback modulation of retinotopic neural activity in targeted areas of the visual cortex (Scharnowski et al., 2012). Similar effects can be also achieved exogenously via rhythmic visual stimulation (Mathewson et al., 2012) and transcranial magnetic stimulation (TMS) (Romei et al., 2012).

Neurofeedback training causes changes in neural activity, which are linked to a shift in the cortical balance of excitation/inhibition (Ros et al., 2010, 2014; Studer et al., 2014). Particularly, suppression of α-activity causes an increment of cortico-spinal excitability and a decrement of intra-cortical inhibition which was the very first evidence of linking neurofeedback modulation to the cortical excitation/inhibition balance (Ros et al., 2010). In order to achieve shifts of excitation/inhibition balance during clinical practice and cognitive tasks via neurofeedback modulation (Ros et al., 2014), cognitive and voluntary demanding approaches should be adopted. A closed-loop stimulation as part of a neurofeedback strategy has an advantage over alternative methods since it combines endo/exogenous methodology such as using specific attributes of neuronal activity that can trigger different sensory stimuli and can finally influence spontaneous activity.

In a recent study, in this issue of Clinical Neurophysiology, Zhigalov and colleagues examined a closed-loop neurofeedback paradigm where high amplitude α oscillatory dynamics trigger flash stimuli during an eyes-closed resting-state task (Zhigalov et al., 2016). The stimulation threshold that was adopted allowed to control the stimulation rate through the adjustment of α oscillatory amplitude via intrinsic neuroregulation. Linking α oscillatory activity with excitability, the adjustments of α oscillatory amplitude via intrinsic neuroregulation are directly connected with shifts in excitation/inhibition balance (Wang, 2010). Additionally, subjects were not aware of the link between visual stimuli and ongoing neuronal activity and for that reason the experimental paradigm can assess the effects of endogenous adaptive mechanisms (Kaplan et al. 2005; Batty et al., 2006). This novel closed-loop paradigm allowed to suppress evoked responses and LRTCs of ongoing brain activity without any significant changes in the α power spectrum. The results based on the estimated scaling exponents of EEG LRTCs during the closed-loop neurofeedback paradigm were compared with a disconnected sham condition. This study presents a proof of concept for a novel closed-loop neurofeedback paradigm that imply changes on the operating point of brain dynamics over the sub/super critical dimensions. This was achieved by mediating the balance between excitation and inhibition via the closed-loop neuroregulation strategy (Zhigalov et al., 2016).

This closed-loop neurofeedback strategy that modulates the LRTC opens new avenues for studying the functional role of brain dynamics and criticality in healthy subjects and for designing novel therapeutic protocols for various brain disorders and diseases that are linked to LRTCs (Zhigalov et al., 2016). Criticality estimated with LRTCs is the universal signature of healthy brain systems that can be analyzed by multichannel recordings from various neuroimaging methods (Massobrio et al., 2015). Experimental observations of LRTCs estimated over phase synchronization in EEG/MEG signals suggest that the main driving mechanism of the observed avalanche activity is global where all temporal scales contribute to the characteristic system behavior (Botcharova et al., 2014). LRTCs have been reported as being impaired in epilepsy (Monto et al., 2007), Alzheimer's disease (Montez et al., 2009), schizophrenia (Nikulin et al., 2012), major depressive disorder (Linkenkaer-Hansen et al., 2005), post-traumatic stress disorder (PTSD) (Ros et al., 2015; for reviews see Cohen et al., 2010) and in age-related cognitive disorders (Mishra and Gazzaley, 2014) and, for that reason, a closed-loop neurofeedback approach could be a valuable tool for non-pharmaceutical treatment (Zhigalov et al., 2016).

Conflict of interest

The author has no potential conflicts of interest to be disclosed.

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